

What is claimed is:

1. An antagonist that inhibits or an agonist that activates an activity a polypeptide selected from the group consisting of: a polypeptide comprising an amino acid sequence which is at least 90% identical to the amino acid sequence of SEQ ID NO:2 or 4,
 - 5 and a polypeptide comprising an amino acid sequence as set forth in SEQ ID NO:2 or 4, wherein said activity is selected from the group consisting of:
 - uncompetitive inhibition by Apo-ACP versus NADH (Kitapp);
 - competitive inhibition by Apo-ACP versus crotonoyl CoA;
 - induction of negative cooperativity with respect to CCA binding;
 - 10 use of NADH and NADPH as substrates by Fab I;
 - binding of NADH and NADPH by FabI;
 - oxidation of NADH and NADPH by FabI;
 - ratio of K_{mapp} for NADH as compared to NADPH;
 - use of NADH and crotonoyl CoA as substrates by Fab I in a sequential
 - 15 kinetic mechanism;
 - sequential binding of NADH and crotonoyl CoA by Fab I;
 - increasing inhibition of FabI by saturated fatty acyl CoA's of increasing chain length;
 - feedback regulatory mechanism of Fab I by saturated fatty acyl CoA's;
 - 20 competitive inhibition by palmitoyl CoA versus crotonoyl CoA;
 - competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation through binding of multiple palmitoyl CoA molecules to Fab I;
 - binding of multiple palmitoyl CoA molecules to Fab I;
 - negative cooperativity in the binding of CCA;
 - 25 formation of an dimeric quaternary structure;
 - formation of an tetrameric quaternary structure;
 - formation of an oligomeric quaternary structure;
 - binding of Fab I by pseudo-product inhibitors beta-NADP+ or palmitoyl coA; and
 - 30 NADH binding to Fab I prior to or simultaneous with ACP binding.
2. A method for the treatment of an individual having need to inhibit or activate Fab I polypeptide comprising the steps of: administering to the individual a antibacterially

effective amount of an antagonist that inhibits or an agonist that activates an activity of a polypeptide selected from the group consisting of: a polypeptide comprising an amino acid sequence which is at least 90% identical to the amino acid sequence of SEQ ID NO:2 or 4, and a polypeptide comprising an amino acid sequence as set forth in SEQ ID NO:2 or 4,

5 wherein said activity is selected from the group consisting of:

uncompetitive inhibition by Apo-ACP versus NADH (K_{iapp});

competitive inhibition by Apo-ACP versus crotonoyl CoA;

induction of negative cooperativity with respect to CCA binding;

use of NADH and NADPH as substrates by Fab I;

10 binding of NADH and NADPH by Fab I;

oxidation of NADH and NADPH by Fab I;

ratio of K_{mapp} for NADH as compared to NADPH;

use of NADH and crotonoyl CoA as substrates by Fab I in a sequential kinetic mechanism;

15 sequential binding of NADH and crotonoyl CoA by Fab I;

increasing inhibition of Fab I by saturated fatty acyl CoA's of increasing chain length;

feedback regulatory mechanism of Fab I by saturated fatty acyl CoA's;

competitive inhibition by palmitoyl CoA versus crotonoyl CoA;

20 competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation through binding of multiple palmitoyl CoA molecules to Fab I;

binding of multiple palmitoyl CoA molecules to Fab I;

negative cooperativity in the binding of CCA;

formation of an dimeric quaternary structure;

25 formation of an tetrameric quaternary structure;

formation of an oligomeric quaternary structure;

binding of Fab I by pseudo-product inhibitors beta-NADP⁺ or palmitoyl coA; and

NADH binding to Fab I prior to or simultaneous with ACP binding.

30 3. A method for the treatment of an individual infected with a bacteria comprising the steps of administering to the individual a antibacterially effective amount of an antagonist that inhibits or an agonist that activates an activity of a polypeptide selected from

the group consisting of: a polypeptide comprising an amino acid sequence which is at least 90% identical to the amino acid sequence of SEQ ID NO:2 or 4, and a polypeptide comprising an amino acid sequence as set forth in SEQ ID NO:2 or 4, wherein said activity is selected from the group consisting of:

- 5 uncompetitive inhibition by Apo-ACP versus NADH (K_{iapp});
- competitive inhibition by Apo-ACP versus crotonoyl CoA;
- induction of negative cooperativity with respect to CCA binding;
- use of NADH and NADPH as substrates by Fab I;
- binding of NADH and NADPH by FabI;
- 10 oxidation of NADH and NADPH by FabI;
- ratio of K_{mapp} for NADH as compared to NADPH;
- use of NADH and crotonoyl CoA as substrates by Fab I in a sequential kinetic mechanism;
- sequential binding of NADH and crotonoyl CoA by Fab I;
- 15 increasing inhibition of FabI by saturated fatty acyl CoA's of increasing chain length; feedback regulatory mechanism of Fab I by saturated fatty acyl CoA's;
- competitive inhibition by palmitoyl CoA versus crotonoyl CoA;
- competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation
- 20 through binding of multiple palmitoyl CoA molecules to Fab I;
- binding of multiple palmitoyl CoA molecules to Fab I;
- negative cooperativity in the binding of CCA;
- formation of a dimeric quaternary structure;
- formation of a tetrameric quaternary structure;
- 25 formation of an oligomeric quaternary structure;
- binding of Fab I by pseudo-product inhibitors beta-NADP⁺ or palmitoyl coA; and
- NADH binding to Fab I prior to or simultaneous with ACP binding.
4. The method of claim 3 wherein said bacteria is selected from the group
- 30 consisting of a member of the genus *Staphylococcus*, *Staphylococcus aureus*, a member of the genus *Streptococcus*, and *Streptococcus pneumoniae*.

5. A method for the treatment of an individual having need to inhibit or activate Fab I polypeptide comprising the steps of administering to the individual a antibacterially effective amount of an antagonist that inhibits or an agonist that activates an activity of Fab I selected from the group consisting of:

- 5 uncompetitive inhibition by Apo-ACP versus NADH (Kitapp);
- competitive inhibition by Apo-ACP versus crotonoyl CoA;
- induction of negative cooperativity with respect to CCA binding;
- use of NADH and NADPH as substrates by Fab I;
- binding of NADH and NADPH by FabI;
- 10 oxidation of NADH and NADPH by FabI;
- ratio of K_{mapp} for NADH as compared to NADPH;
- use of NADH and crotonoyl CoA as substrates by Fab I in a sequential kinetic mechanism;
- sequential binding of NADH and crotonoyl CoA by Fab I;
- 15 increasing inhibition of FabI by saturated fatty acyl CoA's of increasing chain length; feedback regulatory mechanism of Fab I by saturated fatty acyl CoA's;
- competitive inhibition by palmitoyl CoA versus crotonoyl CoA;
- competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation
- 20 through binding of multiple palmitoyl CoA molecules to Fab I;
- binding of multiple palmitoyl CoA molecules to Fab I;
- negative cooperativity in the binding of CCA;
- formation of an dimeric quaternary structure;
- formation of an tetrameric quaternary structure;
- 25 formation of an oligomeric quaternary structure;
- binding of Fab I by pseudo-product inhibitors beta-NADP⁺ or palmitoyl coA; and
- NADH binding to Fab I prior to or simultaneous with ACP binding.

6. A method for the treatment of an individual infected with a bacteria
- 30 comprising the steps of administering to the individual a antibacterially effective amount of an antagonist that inhibits or an agonist that activates that activates an activity of Fab I selected from the group consisting of:

- uncompetitive inhibition by Apo-ACP versus NADH (Kitapp);
- competitive inhibition by Apo-ACP versus crotonoyl CoA;
- induction of negative cooperativity with respect to CCA binding;
- use of NADH and NADPH as substrates by Fab I;
- 5 binding of NADH and NADPH by FabI;
- oxidation of NADH and NADPH by FabI;
- ratio of K_{mapp} for NADH as compared to NADPH;
- use of NADH and crotonoyl CoA as substrates by Fab I in a sequential kinetic mechanism;
- 10 sequential binding of NADH and crotonoyl CoA by Fab I;
- increasing inhibition of FabI by saturated fatty acyl CoA's of increasing chain length; feedback regulatory mechanism of Fab I by saturated fatty acyl CoA's;
- competitive inhibition by palmitoyl CoA versus crotonoyl CoA;
- 15 competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation through binding of multiple palmitoyl CoA molecules to Fab I;
- binding of multiple palmitoyl CoA molecules to Fab I;
- negative cooperativity in the binding of CCA;
- formation of an dimeric quaternary structure;
- 20 formation of an tetrameric quaternary structure;
- formation of an oligomeric quaternary structure;
- binding of Fab I by pseudo-product inhibitors beta-NADP+ or palmitoyl coA; and
- NADH binding to Fab I prior to or simultaneous with ACP binding.
- 25 7. The method of claim 6 wherein said bacteria is selected from the group consisting of: a member of the genus *Staphylococcus*, *Staphylococcus aureus*, a member of the genus *Streptococcus*, and *Streptococcus pneumoniae*.
- 8. A method for the treatment of an individual infected by *Streptococcus pneumoniae* comprising the steps of administering to the individual a antibacterially effective
- 30 amount of an antagonist that inhibits or an agonist that activates an activity of *Streptococcus pneumoniae* Fab I selected from the group consisting of:
 - uncompetitive inhibition by Apo-ACP versus NADH (Kitapp);

- competitive inhibition by Apo-ACP versus crotonoyl CoA;
induction of negative cooperativity with respect to CCA binding;
use of NADH and NADPH as substrates by Fab I;
binding of NADH and NADPH by Fab I;
5 oxidation of NADH and NADPH by Fab I;
ratio of K_{mapp} for NADH as compared to NADPH;
use of NADH and crotonoyl CoA as substrates by Fab I in a sequential
kinetic mechanism;
sequential binding of NADH and crotonoyl CoA by Fab I;
10 increasing inhibition of Fab I by saturated fatty acyl CoA's of increasing
chain length; feedback regulatory mechanism of Fab I by saturated fatty
acyl CoA's;
competitive inhibition by palmitoyl CoA versus crotonoyl CoA;
competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation
15 through binding of multiple palmitoyl CoA molecules to Fab I;
binding of multiple palmitoyl CoA molecules to Fab I;
negative cooperativity in the binding of CCA;
formation of an dimeric quaternary structure;
formation of an tetrameric quaternary structure;
20 formation of an oligomeric quaternary structure;
binding of Fab I by pseudo-product inhibitors beta-NADP⁺ or palmitoyl
CoA; and
NADH binding to Fab I prior to or simultaneous with ACP binding.
9. An antagonist that inhibits an activity of a polypeptide selected from the
25 group consisting of: a polypeptide comprising an amino acid sequence which is at least 90%
identical to the amino acid sequence of SEQ ID NO:2 or 4, and a polypeptide comprising an
amino acid sequence as set forth in SEQ ID NO:2 or 4, wherein said activity is selected from
the group consisting of:
- uncompetitive inhibition by Apo-ACP versus NADH (K_{iapp});
30 competitive inhibition by Apo-ACP versus crotonoyl CoA;
induction of negative cooperativity with respect to CCA binding;
use of NADH and NADPH as substrates by Fab I;

- binding of NADH and NADPH by FabI;
 - oxidation of NADH and NADPH by FabI;
 - ratio of K_{mapp} for NADH as compared to NADPH;
 - use of NADH and crotonoyl CoA as substrates by Fab I in a sequential
 - 5 kinetic mechanism;
 - sequential binding of NADH and crotonoyl CoA by Fab I;
 - increasing inhibition of FabI by saturated fatty acyl CoA's of increasing chain length; feedback regulatory mechanism of Fab I by saturated fatty acyl CoA's;
 - 10 competitive inhibition by palmitoyl CoA versus crotonoyl CoA;
 - competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation through binding of multiple palmitoyl CoA molecules to Fab I;
 - binding of multiple palmitoyl CoA molecules to Fab I;
 - negative cooperativity in the binding of CCA;
 - 15 formation of an dimeric quaternary structure;
 - formation of an tetrameric quaternary structure;
 - formation of an oligomeric quaternary structure;
 - binding of Fab I by pseudo-product inhibitors beta-NADP⁺ or palmitoyl coA; and
 - 20 NADH binding to Fab I prior to or simultaneous with ACP binding.
10. A method for the treatment of an individual having need to inhibit Fab I polypeptide comprising the steps of administering to the individual a antibacterially effective amount of an antagonist that inhibits an activity of a polypeptide selected from the group consisting of a polypeptide comprising an amino acid sequence which is at least 90%
- 25 identical to the amino acid sequence of SEQ ID NO:2 or 4, and a polypeptide comprising an amino acid sequence as set forth in SEQ ID NO:2 or 4, wherein said activity is selected from the group consisting of:
- uncompetitive inhibition by Apo-ACP versus NADH ($K_{\text{i(app)}}$);
 - competitive inhibition by Apo-ACP versus crotonoyl CoA;
 - 30 induction of negative cooperativity with respect to CCA binding;
 - use of NADH and NADPH as substrates by Fab I;
 - binding of NADH and NADPH by FabI;

- oxidation of NADH and NADPH by FabI;
- ratio of K_{mapp} for NADH as compared to NADPH;
- use of NADH and crotonoyl CoA as substrates by Fab I in a sequential kinetic mechanism;
- 5 sequential binding of NADH and crotonoyl CoA by Fab I;
- increasing inhibition of FabI by saturated fatty acyl CoA's of increasing chain length; feedback regulatory mechanism of Fab I by saturated fatty acyl CoA's;
- competitive inhibition by palmitoyl CoA versus crotonoyl CoA;
- 10 competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation through binding of multiple palmitoyl CoA molecules to Fab I;
- binding of multiple palmitoyl CoA molecules to Fab I;
- negative cooperativity in the binding of CCA;
- formation of an dimeric quaternary structure;
- 15 formation of an tetrameric quaternary structure;
- formation of an oligomeric quaternary structure;
- binding of Fab I by pseudo-product inhibitors beta-NADP+ or palmitoyl coA; and
- NADH binding to Fab I prior to or simultaneous with ACP binding.
- 20 11. A method for inhibiting an activity of Fab I polypeptide comprising the steps of contacting a composition comprising said polypeptide with an effective amount of an antagonist that inhibits an activity of Fab I, wherein said activity is selected from the group consisting of:
 - uncompetitive inhibition by Apo-ACP versus NADH (K_{iapp});
 - 25 competitive inhibition by Apo-ACP versus crotonoyl CoA;
 - induction of negative cooperativity with respect to CCA binding;
 - use of NADH and NADPH as substrates by Fab I;
 - binding of NADH and NADPH by FabI;
 - oxidation of NADH and NADPH by FabI;
 - 30 ratio of K_{mapp} for NADH as compared to NADPH;
 - use of NADH and crotonoyl CoA as substrates by Fab I in a sequential kinetic mechanism;

- sequential binding of NADH and crotonoyl CoA by Fab I;
increasing inhibition of FabI by saturated fatty acyl CoA's of increasing
chain length; feedback regulatory mechanism of Fab I by saturated fatty
acyl CoA's;
- 5 competitive inhibition by palmitoyl CoA versus crotonoyl CoA;
competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation
through binding of multiple palmitoyl CoA molecules to Fab I;
binding of multiple palmitoyl CoA molecules to Fab I;
negative cooperativity in the binding of CCA;
- 10 formation of an dimeric quaternary structure;
formation of an tetrameric quaternary structure;
formation of an oligomeric quaternary structure;
binding of Fab I by pseudo-product inhibitors beta-NADP+ or palmitoyl
coA; and
- 15 NADH binding to Fab I prior to or simultaneous with ACP binding.
12. A method for inhibiting an activity of Fab I, wherein said activity is selected
from the group consisting of:
- uncompetitive inhibition by Apo-ACP versus NADH (K_{iapp});
competitive inhibition by Apo-ACP versus crotonoyl CoA;
- 20 induction of negative cooperativity with respect to CCA binding;
use of NADH and NADPH as substrates by Fab I;
binding of NADH and NADPH by FabI;
oxidation of NADH and NADPH by FabI;
ratio of K_{mapp} for NADH as compared to NADPH;
- 25 use of NADH and crotonoyl CoA as substrates by Fab I in a sequential
kinetic mechanism;
sequential binding of NADH and crotonoyl CoA by Fab I;
increasing inhibition of FabI by saturated fatty acyl CoA's of increasing
chain length; feedback regulatory mechanism of Fab I by saturated fatty
acyl CoA's;
- 30 competitive inhibition by palmitoyl CoA versus crotonoyl CoA;

- competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation through binding of multiple palmitoyl CoA molecules to Fab I;
 binding of multiple palmitoyl CoA molecules to Fab I;
 negative cooperativity in the binding of CCA;
 5 formation of an dimeric quaternary structure;
 formation of an tetrameric quaternary structure;
 formation of an oligomeric quaternary structure;
 binding of Fab I by pseudo-product inhibitors beta-NADP⁺ or palmitoyl coA; and
 10 NADH binding to Fab I prior to or simultaneous with ACP binding.
13. The method of claim 12 wherein said bacteria is selected from the group consisting of: a member of the genus *Staphylococcus*, *Staphylococcus aureus*, a member of the genus *Streptococcus*, and *Streptococcus pneumoniae*.
14. A method for inhibiting a growth of bacteria comprising the steps of
 15 contacting a composition comprising bacteria with an antibacterially effective amount of an antagonist that inhibits an activity of Fab I, wherein said activity is selected from the group consisting of:
 uncompetitive inhibition by Apo-ACP versus NADH (Kitapp);
 competitive inhibition by Apo-ACP versus crotonoyl CoA;
 20 induction of negative cooperativity with respect to CCA binding;
 use of NADH and NADPH as substrates by Fab I;
 binding of NADH and NADPH by FabI;
 oxidation of NADH and NADPH by FabI;
 ratio of Kmapp for NADH as compared to NADPH;
 25 use of NADH and crotonoyl CoA as substrates by Fab I in a sequential kinetic mechanism;
 sequential binding of NADH and crotonoyl CoA by Fab I;
 increasing inhibition of FabI by saturated fatty acyl CoA's of increasing chain length; feedback regulatory mechanism of Fab I by saturated fatty
 30 acyl CoA's;
 competitive inhibition by palmitoyl CoA versus crotonoyl CoA;

- competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation through binding of multiple palmitoyl CoA molecules to Fab I;
binding of multiple palmitoyl CoA molecules to Fab I;
negative cooperativity in the binding of CCA;
5 formation of an dimeric quaternary structure;
formation of an tetrameric quaternary structure;
formation of an oligomeric quaternary structure;
binding of Fab I by pseudo-product inhibitors beta-NADP+ or palmitoyl coA; and
10 NADH binding to Fab I prior to or simultaneous with ACP binding.
15. The method of claim 14 wherein said bacteria is selected from the group consisting of: a member of the genus *Staphylococcus*, *Staphylococcus aureus*, a member of the genus *Streptococcus*, and *Streptococcus pneumoniae*.
16. A method for inhibiting a Fab I polypeptide comprising the steps of
15 contacting a composition comprising bacteria with an antibacterially effective amount of an antagonist that inhibits an activity of Fab I, wherein said activity is selected from the group consisting of:
uncompetitive inhibition by Apo-ACP versus NADH (K_{iapp});
competitive inhibition by Apo-ACP versus crotonoyl CoA;
20 induction of negative cooperativity with respect to CCA binding;
use of NADH and NADPH as substrates by Fab I;
binding of NADH and NADPH by FabI;
oxidation of NADH and NADPH by FabI;
ratio of K_{mapp} for NADH as compared to NADPH;
25 use of NADH and crotonoyl CoA as substrates by Fab I in a sequential kinetic mechanism;
sequential binding of NADH and crotonoyl CoA by Fab I;
increasing inhibition of FabI by saturated fatty acyl CoA's of increasing chain length; feedback regulatory mechanism of Fab I by saturated fatty
30 acyl CoA's;
competitive inhibition by palmitoyl CoA versus crotonoyl CoA;

- competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation
- through binding of multiple palmitoyl CoA molecules to Fab I;
- binding of multiple palmitoyl CoA molecules to Fab I;
- negative cooperativity in the binding of CCA;
- 5 formation of an dimeric quaternary structure;
- formation of an tetrameric quaternary structure;
- formation of an oligomeric quaternary structure;
- binding of Fab I by pseudo-product inhibitors beta-NADP+ or palmitoyl
- coA; and
- 10 NADH binding to Fab I prior to or simultaneous with ACP binding.

17. The method of claim 16 wherein said bacteria is selected from the group consisting of: a member of the genus *Staphylococcus*, *Staphylococcus aureus*, a member of the genus *Streptococcus*, and *Streptococcus pneumoniae*.